

# "CHIROPRACTIC MANAGEMENT OF PRIMARY DYSMENORRHEA."

by

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"A Dissertation submitted in partial compliance with the requirements  
for the Masters Degree in Technology in  
the Department of Chiropractic at the Technikon Natal."

I declare that this dissertation represents my own work.

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DATE

# DEDICATION

I dedicate this dissertation to Wayne,  
my husband,  
whose love, support, friendship and guidance  
has been constant.

# ACKNOWLEDGEMENTS

Thank you to our Heavenly Father

for His Divine Protection.

My sincere appreciation go to my parents

without whom none of this would have been possible.

I would also like to thank Dr Till for his guidance and patience.

# ABSTRACT

The purpose of this investigation was to determine the efficacy of chiropractic treatment in the management of primary dysmenorrhea.

The sample consisted of 30 patients who were randomly assigned to 2 groups, the control and experimental.

This single blind study consisted of 16 visits, twice a week for the first 4 weeks and thereafter once a week for the next 8 weeks. During a menstrual cycle, prior to commencement of treatment, the patients were required to complete a Short-Form McGill Pain Questionnaire on the last day of dysmenorrhea and a Numerical Pain Rating Scale 101 on each day of experienced menstrual pain. These questionnaires were completed at home.

Treatment for the experimental group consisted of soft tissue massage of the lumbar and thoraco-lumbar paravertebral musculature combined with spinal manipulative therapy of the areas of fixation in the lumbar and sacro-iliac regions. The control group received purely soft tissue massage of the lumbar and thoraco-lumbar paravertebral musculature. The areas of fixation were determined by motion palpation, joint challenge and tenderness to spinal palpation.

There was no follow-up visit conducted in this study.

An analysis of the data revealed a statistically significant improvement in the experimental group in terms of the Short Form McGill Pain Questionnaire ( $p < 0,001$ ) as well as for the control group ( $p < 0,01$ ), whilst in terms of the Numerical Pain Rating Scale 101 the experimental group showed an improvement ( $p < 0,05$ ) but the control group failed to show a significant change ( $p = 0,068$ ).

When comparing the two groups at the end of the study, in terms of the Short-Form McGill Pain Questionnaire, there was no statistically significant difference ( $p=0,076$ ). When comparing the two groups at the end of the study, in terms of the Numerical Pain Rating Scale 101, there was no statistically significant difference ( $p=0,43$ ).

In respect of this data, spinal manipulative therapy is no more effective than soft tissue therapy.

However, the relatively small sample size may have resulted in a Type II error (i.e. falsely accepting the null hypothesis).

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# CHAPTER ONE

## INTRODUCTION

Dysmenorrhea unrelated to any identifiable disorder almost always begins before twenty years of age and infrequently within the first year after menarche. Primary dysmenorrhea is estimated to affect up to 50% of women of childbearing age, and of these, 10% experience incapacitating pain for 1-3 days every month. It is one of the most important causes of lost working hours and failure to attend school. (Kokjohn, et al. 1992.)

The pain is colicky in nature and is thought to be related to smooth muscle contractions induced by prostaglandins formed in the secretory endometrium and released at the time of endometrial breakdown. (Greenspan. 1991. Page 460.)

When severe, the pain may radiate from the pelvic region to the back and thighs. This may be accompanied by nausea, headaches, vomiting, diarrhoea and emotional disorders. (Greenspan. 1991. Page 459.)

Treatment with Aspirin before the onset of pain is effective in milder cases. However the Non-Steroidal Anti-Inflammatory compounds, which actively inhibit prostaglandin synthesis are more effective when the pain is severe. Naproxin, Ibuprofen, Indomethacin and Mefenamic Acid are more effective if started before the pain begins. (Greenspan. 1991. Page 460.)

As dysmenorrhea accompanies ovulation, suppression of ovulation with oral contraceptives can also be an effective form of treatment. (Greenspan. 1991. Page 460.)

When either oral contraceptives or prostaglandin synthetase inhibitors fail to relieve symptoms after an adequate trial therapy of three to six

months, diagnostic laparoscopy may be indicated. (Wilson and Foster. 1992. Page 762.)

A review of the related literature revealed no data regarding the cost-effectiveness of the various forms of treatment mentioned above. This information might have shown a place for other more conservative and possibly cost-effective interventions such as spinal manipulative therapy.

It is possible that spinal manipulative therapy will reduce the pain and symptoms associated with menstruation and that it may have a reducing effect on plasma prostaglandin levels.

Such a study could demonstrate whether manipulative therapy may be an effective and safe non-pharmacological alternative for relieving the pain and distress of primary dysmenorrhea.

# CHAPTER TWO

## REVIEW OF THE RELATED LITERATURE

## 2.1. DYSMENORRHEA

### 2.1.1. DEFINITION

Primary / Spasmodic Dysmenorrhea is painful menstruation (cramping) for which no organic basis is evident. It usually commences 1 - 2 years after menarche. The pain occurs 12 - 24 hours prior to the onset of menstruation and may last for 1 - 2 days. Cramping is usually located in the lower abdomen. (Jones, et al. 1984. Page 183.)

Dysmenorrhea may be relieved after vaginal delivery of an infant or may continue through much of the woman's reproductive life and then fade, usually during the fourth decade of life. Dysmenorrhea is associated with ovulatory, regular periods and not with anovulatory, irregular menses. (Benson. 1983. Page 659.)

### 2.1.2. AETIOLOGY

Menstrual cramps are thought to be a type of ischaemic pain, that is, when the muscle is made to work with an inadequate blood and oxygen supply, pain results. (Benson. 1983. Page 662.)

Primary dysmenorrhea is attributed to an increased endometrial synthesis of prostaglandin E<sub>2</sub> and F<sub>2α</sub> during endometrial necrosis. The prostaglandins are absorbed into the uterine musculature and blood supply and they mediate the contractions. (Benson. 1983. Page 662.)

The muscle contractions are strong enough to squeeze down on the blood vessels, thereby reducing the blood supply. As long as the

contraction lasts, the uterine muscle has an inadequate supply of blood and oxygen, resulting in severe pain. (Benson. 1983. Page 662.)

Prostaglandin  $F_{2\alpha}$  and  $TXA_2$  are potent vasoconstrictors and may explain dysmenorrhea of obscure origin. They are most likely the cause of the endometrial vasoconstriction that precedes vasodilatation and menstrual bleeding. (Benson. 1983. Page 662.)

A basic organ dysfunction is suggested, as the pain can be relieved by converting the ovulatory cycle to anovulatory cycle with hormone therapy. Uterine hypertonicity and dyskinesia have been noted in association with dysmenorrhea. These signs together with nausea and diarrhoea are described as prostaglandin effects. (Benson. 1983. Page 662.)

Primary dysmenorrhea is also considered to have a psychological component. These psychological factors include tension, anxiety, or using menstrual pain as an attention-getting mechanism. (Benson. 1983. Page 659.)

### 2.1.3. SIGNS AND SYMPTOMS

The lower abdominal pain may be crampy, colicky or a dull constant ache and may extend to the lower back and thighs. It tends to start prior to, or together with, the menses, and may peak after 24 hours. Blood clots can be expelled. (Berkow and Fletcher. 1987. Page 1712.)

### 2.1.4. ASSOCIATED SIGNS AND SYMPTOMS

There may be abdominal distension and heaviness, weight gain, breast swelling and tenderness, nausea with or without vomiting, diarrhoea or



constipation, premenstrual tension, depression, irritability, headache and fatigue. (Hurst. 1988. Page 1121.)

#### 2.1.5. PREVALENCE AND SEVERITY

This condition occurs primarily in adolescent girls and usually subsides by the age of 25 years. It accounts for about 80% of cases of painful menses. Dysmenorrhea is less prevalent and less severe in women with later menarche. It is also less prevalent in women who are parous. The rate of frequency of painful menstruation increases with an increase of chronological age. It is the highest at the age of 16 - 19 years. The prevalence and severity of dysmenorrhea is lower at 25 years of age than at 18 years of age. (Kokjohn, et al. 1992.)

Kokjohn, et al (1992) measured perceived abdominal and back pain by means of a visual analogue scale, and a Menstrual Distress Questionnaire was also used.

#### 2.1.6. DEMOGRAPHICS

Primary dysmenorrhea is estimated to affect up to 50 % of women of child-bearing age and of these, 10 % experience incapacitating pain for 1 - 3 days every month. Due to its recurrent nature, this condition has a great socio-economic impact as well as personal and family consequences. (Kokjohn, et al. 1992.)

#### 2.1.7. TREATMENT

Significantly elevated levels of prostaglandin (PGF<sub>2α</sub>) have been detected in the endometrium and menstrual fluid of primary dysmenorrhea sufferers. These abnormal levels can be reduced and pain

relief achieved with prostaglandin synthetase inhibitors or anti-prostaglandin drugs. (Benson. 1983. Page 662.)

Pain relieving drugs (codeine) or sympathomimetics can be taken to relieve the cramps. If the pain continues to interrupt normal daily activities, low-dose oestrogen - progesterone birth control pills can be taken to suppress ovulation. This is given for only 3 - 4 months and recommenced if the pain returns. (Berkow and Fletcher. 1987. Page 1712.)

Other well-used drugs are the nonsteroidal anti-inflammatories (NSAI) which are prostaglandin synthetase inhibitors. These reduce uterine contractility and discomfort. They are said to be 70 - 85 % effective as pain relievers. A desirable side-effect of the NSAI is the unexplained reduction in blood loss during menstruation. (Kokjohn et al. 1992.)

However, both oral contraceptives and NSAIDs have undesirable side-effects. For example, NSAIDs cause gastro-intestinal disturbances, headache, vertigo and allergic reactions, whilst oral contraceptives are contraindicated in those who wish to fall pregnant or if religious reasons preclude their use. (Kokjohn, et al. 1992.)

The surgical approach to this problem, short of a hysterectomy, involves interruption of the superior hypogastric nerve plexus i.e. a presacral neurectomy, also known as a laparoscopic CO<sub>2</sub> laser uterosacral nerve ablation. This treatment caused a 33 % reduction in linear analogue pain scores. (Benson. 1983. Page 663.)

The literature is not clear in the apparent contradiction in pharmacological and surgical approach to treatment, as referenced in Berkow and Fletcher above.

A paracervical block may be attempted to evaluate the potential effectiveness of a Doyle paracervical neurectomy or a Cott type presacral neurectomy in the patient that is unresponsive to other forms of treatment. Hysterectomy is never indicated as treatment, notwithstanding the fact that 20 - 25 % of patients treated medically for this problem fail to show an improvement. (Benson. 1983. Page 663.)

Normal activity during menstruation should be encouraged, and sports and callisthenics are helpful. (Benson. 1983. Page 663.)

Common forms of self-help include massage of the uterus by applying pressure in to the abdomen just above the pubic symphysis and massaging gently, herbal treatments (for example, raspberry leaf tea), homeopathic remedies, acupuncture, acupressure and shiatsu, aromatherapy and nutritional remedies. (Scambler and Scambler. 1993. Page 47.) However, it is not known whether the clinical efficacy of the above measures have been evaluated.

The use of TENS (Transcutaneous Electrical Nerve Stimulation) has been reported to dramatically improve some women's dysmenorrhea. (Kelly. 1993. Page 22; Kokjohn, et al. 1992.)

Kokjohn, et al (1992) conducted a randomised clinical pilot study comparing treatments in two groups, namely, spinal manipulation and sham manipulation. They measured the circulating plasma levels of prostaglandin  $KDPGF_2\alpha$ , perceived abdominal and back pain and perceived menstrual distress. Twenty-four women were randomly assigned to the spinal manipulation group and twenty-one were assigned to the sham group. A visual analogue scale was used to

measure perceived abdominal and back pain. Both were completed fifteen minutes before and sixty minutes after treatment. Blood samples were collected at the same times to determine the plasma levels of KDPGF<sub>2α</sub> by means of radio immunoassay. Statistical evaluation of the data was accomplished by means of an analysis of covariance and paired Student's t tests. Immediately after treatment, the perception of pain and the level of menstrual distress were significantly reduced by means of the spinal manipulative therapy. This was associated with a significant reduction in plasma levels of KDGP<sub>2α</sub>. A significant and similar reduction in plasma KDGP<sub>2α</sub> also occurred in the sham group, showing that a placebo effect was associated with a single sham intervention.

The control group treatment intervention used by Kokjohn et al. (1992) is understandable as this most closely approximates the manipulation. However the risk of inadvertently giving the manipulation is always present as one may not always have the audible release. Thus there is reason to conduct a study using a different approach to the treatment interventions given.

## **2.2. THE NERVE SUPPLY TO THE FEMALE GENITAL TRACT**

Homewood (1962. Page 236.) quoted D. D. Palmer (the founder of chiropractic) as having said, "In making a diagnosis it should be the chiropractor's business, first of all, to determine the nature of the disease by learning what functions are abnormally performed; what part of the body is affected. Then take into consideration the nerves

which ramify the diseased portion in which they are likely to be impinged upon, and lastly, not the first of all, examine the locality where he has decided the luxation exists which by it's displacement impinges upon those nerves."

Sympathetic, parasympathetic and somatic nerves supply the female reproductive organs.

### 2.2.1. THE AUTONOMIC NERVOUS SYSTEM

Diamond (1976. Page 31.) states that there are 2 major divisions which are anatomically and chemically distinct.

The cranio-sacral outflow, more commonly known as the parasympathetic system, is derived from the brainstem nuclei and sacral segments. Its counterpart is known as the thoraco-lumbar outflow on the basis of its originating cells. This is also known as the sympathetic nervous system. (Tan and Wong. 1990. Page 52.)

#### 2.2.1.1. THE PARASYMPATHETIC SYSTEM

These nerves leave the neuraxis in the 3rd, 7th, 9th and 10th cranial nerves and the 2nd, 3rd and 4th sacral nerves. Even though there is an efferent 2 neuron chain, the parasympathetic ganglia are generally situated close to the target organs. As a result the post-ganglionic fibres are very short. The vagal parasympathetic nerve fibres distribute extensively to the thoracic and abdominal viscera. The sacral fibres supply the lower bowel and the urogenital organs. (Diamond. 1976. Page 33.)

#### 2.2.1.2. THE SYMPATHETIC SYSTEM

The sympathetic nerves emanate from the thoracic and upper two lumbar cordal segments. The chief efferent pathway is via the lumbar splanchnic nerves. (Tan and Wong. 1990. Page 52.)

The efferent impulses reach their target organs along a two neuron chain. The cell bodies of the first or preganglionic neurons lie in the lateral columns of the spinal grey matter. The sympathetic efferent fibres emerge together with the somatic efferent fibres in the anterior nerve root. They then detach from the spinal nerve as the white ramus communicantes. The white ramus communicantes then terminates in one of the sympathetic ganglia which form a chain on the antero-lateral aspect of the vertebral bodies from T<sub>1</sub> - L<sub>2</sub>. This is where the majority of fibres synapse with the second neuron. The second neuron then reaches its destination along a blood vessel or by rejoining one of the adjacent spinal nerves as the grey ramus communicantes. Afferent fibres return to the cord by the white ramus communicantes and the posterior nerve root. (Tan and Wong. 1990. Page 57 and 58.)

The sympathetic chain ganglia are in close anatomical relation to the sides of the vertebral bodies and the costovertebral joints (Tan and Wong. 1990. Page 53). This introduces the possibility that oedema or an inflammatory reaction around those joints could affect the function of the ganglia.

However, the denervated uterus is able to perform normally in parturition, which indicates that the uterus is capable of a significant amount of local autonomy. (Bourdillon, Day, Bookhout. 1992. page 42.)

### 2.2.2. THE HYPOGASTRIC PLEXUS

This plexus is a continuation inferiorly of the aortic plexus and lies in the angle of bifurcation of the aorta between the common iliac arteries. It divides into a right and left pelvic plexus to supply the pelvic viscera. Sympathetic fibres are derived from the first two paravertebral ganglia, the lumbar ganglia and even from the lower thoracic ganglia to join the pelvic plexus. The preganglionic parasympathetic fibres from the 2nd and 3rd sacral nerves join the pelvic plexus of the same side. (Moore. 1985. Page 388.)

The visceral structures supplied by the subdivisions of the pelvic plexus are the rectum, urinary bladder, prostate gland, seminal vesicles, testicle and penis of the male and uterus, vagina and ovary of the female. (Moore. 1985. Page 388.)

### 2.2.3. INNERVATION OF THE ORGANS

The fallopian tubes and uterus receive sympathetic preganglionic fibres which arise in T<sub>10</sub> - L<sub>2</sub> spinal cord segments, pass through the hypogastric plexus and the utero-vaginal plexus in the broad ligament. They then synapse in these plexuses which contain both sympathetic and parasympathetic fibres. (Moore. 1985. Page 375.)

The nerve supply to the vagina is via the pudendal and haemorrhoidal nerves and the pelvic sympathetic chain. Innervation of the cervix is via the 2nd, 3rd and 4th sacral nerves and the pelvic sympathetic plexus. (Benson. 1983. Page 16.)

Afferent pain impulses from the cervix and uterus are carried by nerves that accompany sympathetic fibres and enter the neuraxis at T<sub>10</sub> - L<sub>1</sub>. (Appenzeller. 1976. Page 283.)

The sensory nerves from the uterine fundus accompany the sympathetic fibres to the ganglia of T<sub>11</sub> and T<sub>12</sub> posterior roots. These fibres are thought to carry impulses associated with pain.

The parasympathetic supply to the uterus arises from S<sub>2</sub> - 4. The fibres synapse in the utero-vaginal plexus and in the uterine wall. The cervical ganglion, situated on either side of the cervix contains postganglionic, parasympathetic and sympathetic neurons. The parasympathetic fibres are accompanied by sensory fibres that supply the cervix and originate in the sacral spinal ganglia. These fibres convey the sensation of pain from the cervix and this pain is referred to the sacral region. (Appenzeller. 1976. Page 284.)

The autonomic nerve supply is thought to be primarily concerned with the innervation of blood vessels although termination of postganglionic fibres on the uterine muscle have been demonstrated. (Appenzeller. 1976. Page 284.)

#### 2.2.4. THE VENTRAL RAMI

The ventral rami of the lumbar spinal nerves emanate from the intervertebral foramina by penetrating the ventral leaf of the intertransverse ligament, where the changes associated with a subluxation may contribute to nerve root compression syndromes. Accordingly, they enter the space in front of the spinal ligaments, and lie within the psoas major muscle, where muscle contraction could possibly affect them. Within the muscle they enter into the formation



lie within the psoas major muscle, where muscle contraction could possibly affect them. Within the muscle they enter into the formation of plexuses. The lumbar plexus is formed with the ventral rami from L 1 -4 segments and the L 4 - 5 ventral rami join to form the lumbo-sacral trunk which enters the lumbo-sacral plexus. (Bogduk and Twomey. 1991. Page 114.)

#### 2.2.5. CENTRAL TRANSMISSION OF PAIN

The afferent fibres that convey nociception are group A-delta and group C fibres. These fibres enter the dorso-lateral tract of Lissauer located at the tip of the dorsal horn. Within this region collateral branches of these fibres that continue directly into the grey matter ascend or descend several cord segment levels before they enter the dorsal horn. The A-delta fibres, which convey pain rapidly, terminate in lamina II. The neurons which transmit the information to higher centres, are located in various laminae of the grey matter. The major fibres that transmit nociception to higher centres decussate in the ventral white commissure and then ascend in the anterolateral quadrant of the white matter of the spinal cord. (Bourdillon, Day, Bookhout. 1992. Page 42.)

#### 2.2.6. THE NEOSPINOHALAMIC TRACT

One of the tracts in the anterolateral quadrant is the neospinothalamic tract. This tract ascends through the brainstem to the ventral lateral nucleus (posterior part) and also to the posterior nucleus of the thalamus with little or no input to the brainstem from the thalamus. Axons course to the somesthetic region of the cortex, that is, the post-central gyrus and the posterior part of the paracentral

lobule of the parietal lobe. As the axons ascend, body parts are represented in specific regions of the tract, and in the cerebral cortex a pattern is retained such that a specific area of cortex responds to the region of the body from which the sensory fibres originate. This cortical representation is referred to as the sensory homunculus. The size of the homunculus reflects the amount of sensory innervation devoted to that body area. This unequal neuronal representation may explain why localisation of sensations, such as pain, is more difficult in one region than in another. The neospinothalamic tract synapses in the region of the sensory homunculus and provides the base for the discriminatory qualities of pain sensation, such as stimulus intensity and spatial localisation. (Bourdillon, Day, Bookhout. 1992. Page 42.)

#### 2.2.7. THE PALEOSPINOThALAMIC TRACT

An additional tract that ascends in the antero-lateral quadrant is the paleospinothalamic tract. This ascends through the brainstem, and likely contributes collateral branches to the reticular formation, terminates in the midline and intralaminar thalamic nuclei. From these nuclei, thalamic fibres travel to regions associated with the limbic system and to widespread areas of the cerebral cortex such as the orbitofrontal region. (Bourdillon, Day, Bookhout. 1992. Page 43.)

The perception of pain takes place in the thalamus, postcentral gyrus, frontal cortex (affective component) and the temporal cortex (memory of previous pain component). The unpleasant emotional response associated with pain, seems to be associated with the limbic system. This limbic system allows one to perceive a sensation as being uncomfortable, aching or hurting. (Gatterman. 1995. 31 and 32.)

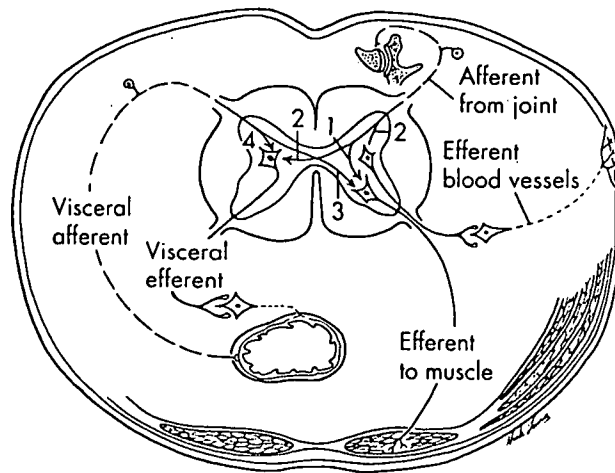
### **2.3. THE NEURODYNAMICS OF THE VERTEBRAL SUBLUXATION**

A visceromotor reflex is established when the prostaglandins released during endometrial necrosis stimulates the receptor nerve endings and send impulses back over the splanchnic afferent fibres to the spinal cord segments. The synapse with the anterior horn cells causes a somatic muscle hypertension involving the postventral muscle fibres of the relevant portion of the spine. (Bergmann, Peterson and Lawrence. 1993. Page 152.)

Afferent fibres synapse with intercalated neurons which eventually link up with the dendrites of the lateral and anterior horn cells at the relevant segmental entrance (Figure 1). The efferent neurons must conduct impulses centrifugally in response to this afferent source of stimulation. To every adequate stimulus there must be a somatic or visceral structural response, as neural impulses are not just stored in the brain and spinal cord. (Bergmann, Peterson and Lawrence. 1993. Page 152.)

The response to this efferent stimulation is likely to be multisegmental and results in increased tonus of the posterior vertebral musculature. The cell bodies of the axons in the anterior grey horns are traceable through the anterior nerve root, through the nerve trunk and the posterior primary ramus of the spinal nerves to the posterior vertebral musculature. The greatest hypertonicity would be observed in the deep segmental muscles, such as the Rotatores and Multifidus muscles. (Homewood. 1962. Page 64.)

Figure 1 - Afferent and efferent pathways from and to the viscera and somatic structures that can produce (1) somatosomatic, (2) somatovisceral, (3) viscerosomatic, and (4) viscerovisceral reflex phenomena. (Bergmann, Peterson and Lawrence. 1993. Page 153.)



### 2.3.1. THE VERTEBRAL SUBLUXATION

A disturbance of normal functioning (fixation) of a vertebral segment is visualised as a disrelation of a vertebral segment in combination with a consecutive vertebra/e. The fixation does not materially alter the dimensions of the intervertebral foramina, yet the disrelation and disturbed function does have consequences for the neuromere and the nerve trunk. (Homewood. 1962. Page 150.)

The skilled chiropractor is proficient in detecting a vertebral segment which is incapable of moving through its full range of motion with the use of motion palpation techniques. Palpatory indicators of a subluxation include increased muscular tonicity within the deeply

seated segmental muscles. Other evidence of a fixation is acute spinous tenderness to palpation. There is difficulty in determining, on a neural basis only, the exact location of a subluxation in any symptom complex. (Homewood. 1962. Page 151.)

#### 2.4. THE NEUROBIOLOGIC MECHANISMS IN MANIPULATIVE THERAPY AND THE NEUROBIOLOGIC HYPOTHESIS

The professions of chiropractic, osteopathy and other "manual medicines" have foreseen manual "hands on" therapy neurologically affecting somatic and visceral disorders. (Bergmann et al. 1993.)

Manipulative therapy entails the application of specifically 'directed manual forces to the body, with the objective of improving mobility in areas of restriction. These areas could be within the joints, connective tissues or in the skeletal muscles. The outcomes may include the improvement of posture and locomotion; pain and discomfort relief; functional improvement elsewhere in the body and improvement of the sense of well-being. (Korr. 1978. Page 219.)

It seems improbable that nerve root compression will be induced by an uncomplicated subluxation. However, it is plausible that pathologic or degenerative changes associated with the subluxation may contribute to nerve root compression syndromes. In these circumstances, Triano states in Haldeman (1992. Page 252.) that adjustive therapy, which reduces the position of a fixed subluxation and nerve root irritation

might cause a reduction in nerve root traction, compression or inflammation.

Somatic dysfunction and joint dysfunction induces persistent nociceptive and altered proprioceptive input. This is known as the impulse based paradigm. A segmental cord response is triggered by the continuous afferent input which in turn generates a pathologic somatosomatic or somatovisceral disease reflex (facilitation). Again, Triano states in Haldeman (1992. Page 252.) that if these reflexes continue, they are assumed to generate altered function in segmentally supplied somatic or visceral structures.

Triano asserts in Haldeman (1992. Page 252.) that by normalising joint mechanics and eliminating the transformed neurogenic reflexes associated with joint dysfunction, there is potential for terminating local and distant somatic and visceral effects. It is theorised that this can be accomplished with chiropractic manipulative techniques.

Triano further states in Haldeman (1992. Page 252.) that this paradigm has become the focus of more attention and inquiry as the chiropractic profession searches for justification to the wide ranging physiologic effects that have been clinically observed to be associated with spinal manipulative therapy.

This relationship, however, is not consistent, and the frequency of response is undetermined, but the anecdotal and empirical experiences of the profession are significant enough to warrant serious further investigation. (Bergmann et al. 1993. Page 118.)

There is also the potential for direct mechanical irritation of the autonomic nervous system. This reasoning is based on the anatomic

proximity and vulnerability of the posterior ganglionated chain situated between the vertebral levels of T<sub>1</sub> and L<sub>2</sub> to the soma of the posterior chest wall and the costovertebral joints. (Bergmann et al. 1993. Page 118.)

It is postulated that segmental sympathetic hypertonia may be induced by altered spinal and costovertebral mechanics which directly irritate the sympathetic ganglia. As a consequence of altered sympathetic function, the target organs within the segmental distribution then become sensitive to the modified autonomic regulation and function. (Bergmann et al. 1993. Page 118.)

In comparison to the sympathetic chain, the parasympathetic system does not have anatomic proximity to the spinal joints. Cervical and pelvic dysfunctional mechanics are possible sources of entrapment or tethering of the parasympathetic fibres which may lead to dysfunction. Dysfunction of the cervical, or cranio-sacral mechanics is hypothesised to induce traction of dural attachments and the cranial nerves as they exit through the dura and skull foraminae. (Bergmann et al. 1993. Page 118.)

The objective of treatment in a mechanically-induced autonomic dysfunction is to detect the locations of joint dysfunctions and implement pertinent manual therapy to stabilise membranous tension. (Bergmann et al. 1993. Page 118.)

When the nervous transmission to the female reproductive organs is altered by fixation in the lower back and sacral area, it is hypothesised that functional disorders may occur in those areas of nervous distribution. The myometrium of the uterus and other muscles in the

area may then be influenced by "facilitated" nerves. Also, the circulation to the associated viscera may be impaired from aberrant sympathetic reflexes. By correcting the joint fixations, the chiropractor may re-establish appropriate nerve transmission to the autonomic nervous system and restore proper nervous transmission to the uterus. The circulatory, visceral and muscular innervation may then be restored and pain alleviated.

From the discussion of spinal dysfunction and its possible neurobiologic effects on health, it must be remembered that spinal dysfunction may be the product of, not the cause of, somatic or visceral dysfunction or disease. Spinal pain and dysfunction may be secondary to a disorder that needs direct treatment. Manual therapy may be a suitable element of appropriate care but would be insufficient as the sole treatment in such a case.



# CHAPTER THREE

MATERIALS AND

METHODS

The subjects were recruited from the local community by means of advertisements, posters or referrals.

Consecutive sampling was employed to select subjects for this study. With this sampling method every subject that met the eligibility requirements was invited to participate in the study.

The sample size consisted of thirty patients. These women were randomly assigned to two groups of fifteen each, namely, the control group and the experimental group. The procedure for randomisation consisted of thirty slips of folded paper, fifteen with Group 1 written on it and fifteen with Group 2 written on it, put into a container from which each subject withdrew one at the beginning of the study. The subjects were not permitted to look at these pieces of paper. Group 1 consisted of the experimental subjects and Group 2 consisted of the control subjects.

The diagnostic criteria for Primary Dysmenorrhea are contained within its definition in the literature review.

The initial consultation included obtaining a signed consent form (Appendix A), a case history (Appendix B), physical examination (Appendix C) and a lumbar spine regional examination (Appendix D). The patients eligible for the study were then required to undergo an internal pelvic examination by a registered gynaecologist to exclude the possibility of underlying pathology. If concurrent myofascial trigger points were found the subjects were still included in the study. Travell and Simons (1983, Page 14.) state that pain from myofascial trigger point syndromes are rarely completely symmetrical. Dysmenorrhea occurs over the entire lower abdomen, and thus it is unlikely that the

two conditions could have been mistaken in diagnosis. However the dysmenorrhea and associated emotional distress, could have indirectly initiated the myofascial trigger points. (Travell and Simons. 1983. Page 14.)

No illnesses were developed by the subjects during the study and no medication was taken by the subjects for associated signs and symptoms of dysmenorrhea (for example, Aspirin for headaches).

The only exclusion criteria were that the subjects had no contraindications to spinal manipulation. The study included women on medication (for example, birth control pills) and this medication was required to remain constant throughout the length of the study.

At the initial consultation the patient received a Numerical Pain Rating Scale 101 (Appendix E) (Jenson et al. 1986.) and a Short-Form McGill Pain Questionnaire (Appendix F) (Melzack. 1987.) to complete during the subsequent menstrual cycle - this was used to record the base pain level. The Numerical Pain Rating Scale 101 was used to measure the intensity of the pain experienced, whilst the Short-Form McGill Pain Questionnaire recorded the character of the pain experienced. The questionnaires were taken home by the subjects to be completed at the end of each pain day.

The study consisted of sixteen visits, twice weekly for the first four weeks and once weekly for the following eight weeks. No follow-up consultation was included.

The control group received soft tissue massage of the para-spinal musculature.

An effleurage massage was given beginning with light pressure progressing to deep stroking. The strokes were done using the palmar surface of the hand, fingers and thumb, keeping in contact with the skin and moving gently over the underlying tissues. These strokes were conducted in a rhythmic, slow fashion. Technical oil (cosmetic oil) was used as a lubricating medium. (Basmajian. 1985. Pages 265 and 268.)

The strokes were executed in a slow circular movement starting from below and moving upward (centripetally) parallel to the long axis of the spine. (Beard and Wood. 1964. Page 42.)

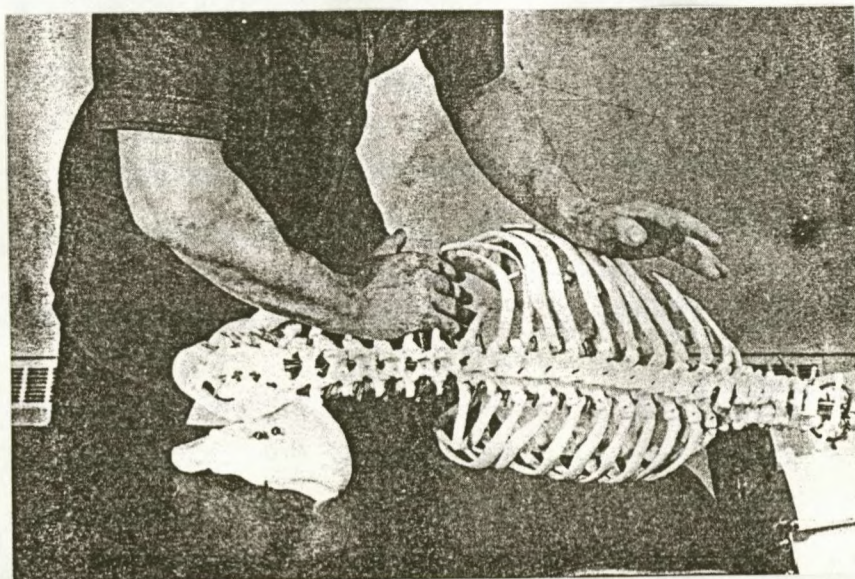
The experimental group received soft tissue massage of the para-spinal musculature as above followed by manipulation of the areas of fixation in the lower thoracic spine, lumbar spine and sacroiliac joints. The most common areas adjusted were L5 and the sacro-iliac joints.

The manipulation was conducted with the patient lying laterally recumbent with the involved side uppermost. The patient's lower shoulder was anterior with the lower forearm flexed and resting on the anterior thoracic wall and the upper shoulder posterior with the forearm flexed and resting on the lateral thoracic wall. The lower thigh and leg were remained straight whilst the upper thigh and leg were flexed with the dorsum of the foot in the popliteal space of the lower limb. The pelvis was rolled anterior on the involved side. The therapists position was anterior to the patient in fencer stance facing cephalad, bending well over the patient with the caudad foot off the floor. Caudad (contact) hand with a base of hand contact on the upper or lower sacro-iliac or a pisiform contact on the mamillary process of the superior lesion with the forearm at 90 degrees to the contact hand.



Cephalad (indifferent) hand with a palmar contact on the anterior aspect of the patient's upper shoulder. The indifferent hand stabilised the patient while the contact hand drove the transverse or mamillary process or sacrum anteriorly and superiorly with a simultaneous body drop. (Figure 2) (Szaraz. 1984. 9.1)

Figure 2 - The position assumed by therapist and patient during the chiropractic manipulation.



Areas of fixation were determined by three palpation techniques:

1. - Spinous tenderness to pressure applied by the examiner's finger. A subjective sign of pain is helpful to the examiner as a confirmation of objective findings at the same level. (Bourdillon, Day and Bookhout. 1992. Page 49.)
2. - Joint challenge (lateral spinous process pressure) (Figure 3). With the patient prone the examiner, using her thumbs, pushed the spinous processes of two consecutive vertebrae laterally away from the midline in opposing directions. Alternating from the left to the right. This test produces forced rotation of contiguous vertebrae. The normal capsular end-feel is "springy" and painless. (Burn. 1994. Page 55.)

Figure 3 - Lateral Spinous Process Pressure (Burn. 1994. Page 59.)



3. - Motion palpation was used to determine the range and quality of joint movement. There was a difference in the "end feel", that is the feel of increasing resistance to motion as the limit is reached. (Bourdillon, Day and Bookhout. 1992. Page 51.)

Motion palpation methods are used to determine the joints in dysfunction and the direction of motion loss. The level of joint dysfunction and the direction in which the joint fails to move, determines the level of the manipulation and the line of drive of a manipulation force. The manipulation is usually directed into the resistance.

Asymmetry is common when dealing with the axial skeleton. Structural asymmetry must be distinguished from dysfunctional asymmetry. Whilst a dysfunctional joint will have some restriction in motion, in the absence of dysfunction a joint with structural asymmetry will have normal mobility. (Bourdillon, Day and Bookhout. 1992. Page 49.)

No physiotherapy, nutritional counselling or other adjunctive therapies were given.

This was a single blind study in that the patients did not know to which group they had been assigned, yet the researcher knew. An objective observer was not used as the assessments were all in the form of questionnaires.

All subjects that were accepted for the study were able to follow the study through to completion.

The Numerical Pain Rating Scale 101 and Short-Form McGill Pain Questionnaires were completed by the subjects during three successive menstrual cycles throughout the treatment period. The data obtained from the patients' questionnaires were analysed by means of the Wilcoxin Signed Rank Test for intragroup comparison (paired data) and the Mann-Whitney U Test for intergroup comparison (unpaired data) with the assumption that both sample groups were from approximate normal distributions and that these distributions had the same variance.



# CHAPTER FOUR

## RESULTS

#### **4.1. CRITERIA FOR ADMISSIBILITY OF THE DATA**

Only the data from the Short-Form McGill Pain Questionnaire and the Numerical Pain Rating Scale 101 that were completed and under supervision were used.

Both the experimental and control groups underwent the same tests and procedures, differing only in the therapeutic intervention.

#### **4.2. DATA OBTAINED IN THE STUDY**

The first questionnaires were completed before therapeutic intervention began. The ensuing two were administered during treatment and the last on completion of the treatments. Thus there are four sets of data for each subject.

TABLE 4.2.1.a.

DATA OBTAINED FROM THE EXPERIMENTAL GROUP

SHORT FORM MCGILL PAIN QUESTIONNAIRE				
PATIENT NUMBER	FIRST CYCLE	SECOND CYCLE	THIRD CYCLE	FOURTH CYCLE
1	13	7	4	2
2	3	1	2	2
3	10	7	8	4
4	25	35	20	7
5	9	12	2	0
6	7	0	5	3
7	7	7	5	6
8	4	5	4	6
9	4	4	1	2
10	10	7	0	6
11	6	4	5	5
12	3	3	3	2
13	31	24	26	22
14	12	5	2	0
15	4	4	3	2
MEAN	9.87	8.33	6	4.6
STANDARD DEVIATION	8.11	9.29	7.26	5.3

TABLE 4.2.1.b.

DATA OBTAINED FROM THE EXPERIMENTAL GROUP

NUMERICAL PAIN RATING SCALE 101				
PATIENT NUMBER	FIRST CYCLE	SECOND CYCLE	THIRD CYCLE	FOURTH CYCLE
1	95	85	70	40
2	75	45	45	50
3	80	75	80	90
4	75	80	70	60
5	80	50	30	0
6	35	0	30	35
7	60	65	60	65
8	60	85	60	70
9	100	80	50	70
10	60	55	0	40
11	70	80	60	75
12	80	60	50	30
13	90	90	80	80
14	98	70	50	0
15	85	65	70	40
MEAN	76.2	65.67	53.67	49.67
STANDARD DEVIATION	17.39	22.67	21.42	26.96

TABLE 4.2.2.a.

## DATA OBTAINED FROM THE CONTROL GROUP

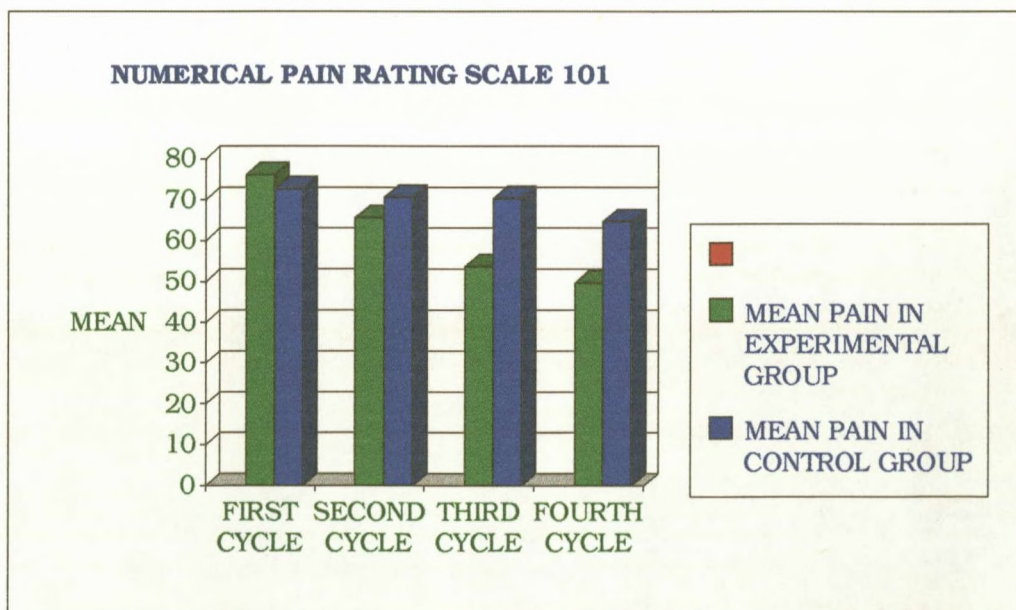
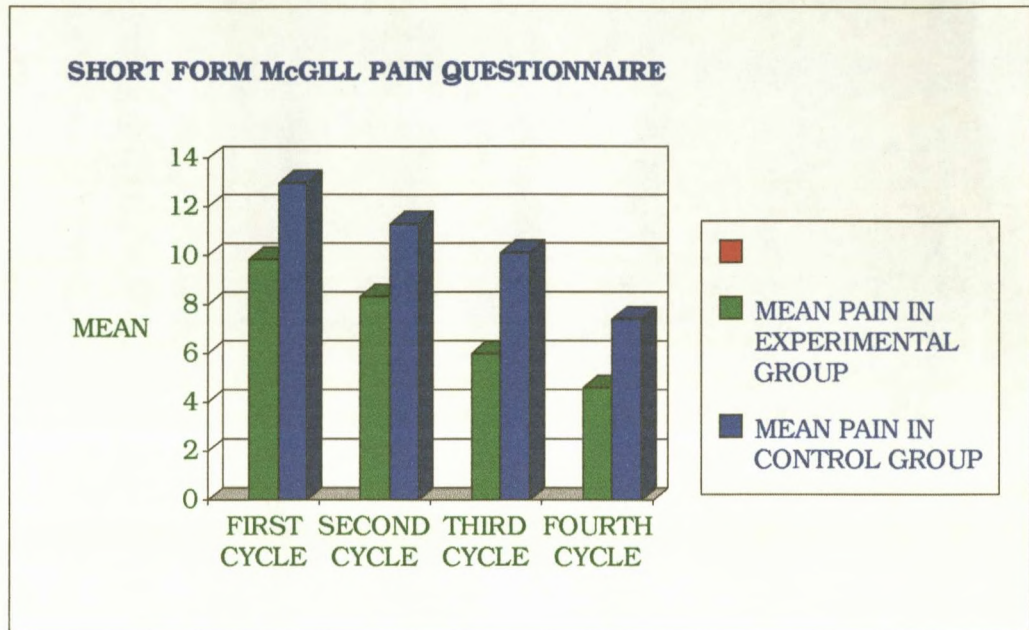
SHORT FORM MCGILL PAIN QUESTIONNAIRE				
PATIENT NUMBER	FIRST CYCLE	SECOND CYCLE	THIRD CYCLE	FOURTH CYCLE
1	6	3	2	1
2	22	20	17	15
3	11	8	9	7
4	10	9	4	2
5	10	12	18	6
6	21	24	10	3
7	18	19	8	10
8	5	6	5	5
9	24	14	14	12
10	6	3	4	5
11	26	5	22	2
12	5	3	4	2
13	10	7	11	6
14	8	9	7	6
15	13	28	17	29
MEAN	13	11.33	10.13	7.4
STANDARD DEVIATION	7.28	8.01	6.17	7.16

TABLE 4.2.2.b.

## DATA OBTAINED FROM THE CONTROL GROUP

NUMERICAL PAIN RATING SCALE 101				
PATIENT NUMBER	FIRST CYCLE	SECOND CYCLE	THIRD CYCLE	FOURTH CYCLE
1	60	40	30	20
2	46	50	40	35
3	80	70	70	70
4	80	60	55	60
5	60	80	90	90
6	75	100	90	55
7	100	100	90	100
8	40	50	30	40
9	85	60	80	70
10	80	50	80	50
11	90	80	100	60
12	50	50	50	50
13	85	80	80	80
14	100	100	90	90
15	60	90	80	100
MEAN	72.73	70.67	70.33	64.67
STANDARD DEVIATION	18.97	20.86	23.34	24.09

GRAPH 4.2.1. REPRESENTING THE MEAN PAIN VALUES  
FROM BOTH THE EXPERIMENTAL AND CONTROL GROUPS



OVERALL OUTCOME OF SUBJECTIVE TESTING AT THE END OF THE STUDY

TABLE 4.2.3.a.

SHORT FORM MCGILL PAIN QUESTIONNAIRE

	PATIENTS' PAIN DECREASED	PATIENTS' PAIN STAYED THE SAME	PATIENTS' PAIN INCREASED
EXPERIMENTAL GROUP	10	1	4
CONTROL GROUP	10	3	2

TABLE 4.2.3.b.

NUMERICAL PAIN RATING SCALE 101

	PATIENTS' PAIN DECREASED	PATIENTS' PAIN STAYED THE SAME	PATIENTS' PAIN INCREASED
EXPERIMENTAL GROUP	14	0	1
CONTROL GROUP	13	1	1



### 4.3. REPORTED DATA

Interesting observations were recorded, on the questionnaires, by two of the subjects in the experimental group. One subject observed an absence of breast swelling and tenderness in the third and fourth cycle, whilst another perceived pain of a shorter time span in her fourth cycle.

The ages of the subjects ranged from 16 years of age to 28 years of age with an equal distribution in both groups. Only one subject in the experimental group was parous. None of the subjects had previous surgery or serious illnesses.

Of the thirty subjects in the study ten were found to have associated myofascial trigger points. The three muscles involved were the Quadratus Lumborum, Gluteus Medius and Piriformis and were found equally distributed in the two groups.

On examination, at the beginning of each treatment, it was found that the subjects had generally a combination of sacro-iliac and lumbar facet syndromes. It was also observed that at some treatments the patients had neither syndromes. As the study spanned three months these patients could have recovered or the conditions could have recurred.

In regard to the contraceptive pill, eighteen patients were on prescriptions for low dose oestrogen - progesterone pills. Only two of these were taking the pill to provide relief for dysmenorrhea. Of the eighteen, eleven were in the experimental group and four in the control group.

#### 4.4. STATISTICAL METHODS USED FOR ANALYSIS OF THE DATA

The hypothesis tests performed on the data gathered from the Short-Form McGill Pain Questionnaire and the Numerical Pain Rating Scale 101, were the Wilcoxin Signed Rank Test and the Mann Whitney U Test. These are non-parametric hypothesis tests. The Wilcoxin Signed Rank Test was used to compare data within the same group whereas the Mann Whitney U Test was utilised to compare data between two different groups. These two tests were conducted at a 5% level of significance.

Eight statistical tests were conducted. The first four were conducted on the results of the Short-Form McGill Pain Questionnaire. The first two of these collated data from the first and the last questionnaires in the experimental and control groups. The third, compared the data from the first questionnaires of the experimental and control groups. The fourth included data from the last questionnaires of the experimental and control groups.

The last four tests were applied to the results of the Numerical Pain Rating Scale 101. The fifth and sixth compared the results of the first and last questionnaires of the experimental and control groups. The seventh test utilised the results of the first questionnaires of the experimental and control groups. The eighth, the results of the last questionnaires of the experimental and control groups.

#### 4.4.1. INTRAGROUP COMPARISON

The intragroup comparison was completed to demonstrate whether there was a statistically significant improvement in each of the two groups. This was done by testing whether the mean pain, as measured by the Short-Form McGill Pain Questionnaire and the Numerical Pain Rating Scale 101, was decreased.

##### Wilcoxin Signed Rank Test

The above statistical inferential test, is applied in the following manner on the data accumulated from the Short-Form McGill Pain Questionnaire and the Numerical Pain Rating Scale 101:

The null hypothesis is stated as:

$$H_0: u_e - u_c = 0$$

(No true difference exists between the  
study groups)

The alternative hypothesis is stated as:

$$H_1: u_e - u_c > 0$$

(There is a true difference between the  
two groups)

The above test is used to test whether both (or any one of) the control and experimental groups showed a statistically significant improvement.

## RESULTS

### 4.4.1.1. SHORT-FORM MCGILL PAIN QUESTIONNAIRE

Experimental group:

Large sample test statistic  $Z = 3.09539$

One tailed probability of equalling or exceeding  $Z = 0.00098$

Control group:

Large sample test statistic  $Z = 2.57384$

One tailed probability of equalling or exceeding  $Z = 0.00503$

### 4.4.1.2. NUMERICAL PAIN RATING SCALE 101

Experimental group:

Large sample test statistic  $Z = 2.07162$

One tailed probability of equalling or exceeding  $Z = 0.01915$

Control group:

Large sample test statistic  $Z = 1.49048$

One tailed probability of equalling or exceeding  $Z = 0.06805$

### 4.4.2. INTERGROUP COMPARISON

This was executed to test whether there was a statistically significant difference in improvement between the 2 groups. This was accomplished by testing whether the mean "pain" of the experimental group was statistically less significant than that of the control group.

The testing procedure used was:

### Mann-Whitney U Test

The above statistical inference test, was applied to the following hypothesis:

$$H_0: u_e = u_c$$

(The mean "pain" of the 2 groups was the same)

$$H_1: u_e < u_c$$

(Mean "pain" in experimental group was less  
than that in the control group)

## RESULTS

### 4.4.2.1. SHORT-FORM MCGILL PAIN QUESTIONNAIRE

Experimental group VS Control group (First questionnaire):

Large sample test statistic  $Z = 1.47774$

One tailed probability of equalling or exceeding  $Z = 0.06974$

Experimental group VS Control group (Fourth questionnaire):

Large sample test statistic  $Z = 1.43045$

One tailed probability of equalling or exceeding  $Z = 0.07629$

#### 4.4.2.2. NUMERICAL PAIN RATING SCALE 101

Experimental group VS Control group (First questionnaire):

Large sample test statistic  $Z = -1.35347$

One tailed probability of equalling or exceeding  $Z = 0.08795$

Experimental group VS Control group (Fourth questionnaire):

Large sample test statistic  $Z = -0.188335$

One tailed probability of equalling or exceeding  $Z = 0.42531$

# CHAPTER FIVE

## DISCUSSION

## 5.1. INTERPRETATION OF THE RESULTS

### 5.1.1. INTRAGROUP COMPARISON

#### Wilcoxin Signed Rank Test

##### 5.1.1.1. SHORT-FORM MCGILL PAIN QUESTIONNAIRE

Experimental group (comparison between the first and fourth questionnaire):

If the one tailed probability of equalling or exceeding Z (0.00098) is less than 0.05 then the null hypothesis was rejected. This meant that there was indeed a statistically significant difference in the mean pain between the first and final menstrual cycles. In other words the experimental group's mean pain did decrease in terms of the Short-Form McGill Pain Questionnaire.

Control group (comparison between the first and fourth questionnaire):

The exceedance probability was 0.00503 which was less than the 5% level of significance. The null hypothesis was rejected which meant that there was a statistically significant difference in the mean pain between the first and last consultations within the control group. This



is to say that, the control group's mean pain decreased in terms of the Short-Form McGill Pain Questionnaire.

The fact that the control groups' mean pain decreased was not expected, can be understood when considering the "placebo response". The treatment given, that is, soft tissue massage, could have initiated a psychoneurophysiological influence on the subjects, bringing about an effect in the body of the patient. However, this is also applicable to the experimental group. Basmajian (1985. Page 105.) stated that a success rate of 30 - 50 % may be achieved during treatment if the therapist appears confident and comes in contact with the patient. The important element seems to be close contact between the patient and the therapist.

#### 5.1.1.2. NUMERICAL PAIN RATING SCALE 101

Experimental group (comparison between the first and fourth questionnaire):

As the exceedance probability (0.01915) was  $< 0.05$ , the null hypothesis was rejected and it was concluded that there was a statistically significant difference in the mean pain between the first and last consultations within the experimental group. As per application of the Numerical Pain Rating Scale 101, a decrease in the experimental group's mean pain was observed.

Control group (comparison between the first and fourth questionnaires):

The exceedance probability here was 0.06805 which was greater than the 5% level of significance. This meant that the null hypothesis was accepted and it was deduced that there was no statistically significant difference in the mean pain between the first and last consultations within the control group.

The difference which seems conflicting between the Short-Form McGill Pain Questionnaire and the Numerical Pain Rating Scale 101, was due to the nature of the two questionnaires. The Short-Form McGill Pain Questionnaire determines the character of the pain experienced while the Numerical Pain Rating Scale 101 ascertained the level of pain experienced.

The possibility of falsely accepting the null hypothesis (a Type II Error) could occur, thus concluding that no difference exists between the mean pain of the first and final consultations within the control group, when in fact there was an actual difference.

#### 5.1.2. INTERGROUP COMPARISON

There was a 26.53 % reduction in mean pain in the Numerical Pain Rating Scale 101 in the experimental group. This compares favourably with the 33 % reduction in linear analogue pain scores with laparoscopic CO<sub>2</sub> laser uterosacral nerve ablation. The control group

showed a 8.06 % reduction in mean pain in the Numerical Pain Rating Scale 101.

#### Mann-Whitney U Test

##### 5.1.2.1. SHORT-FORM MCGILL PAIN QUESTIONNAIRE

Experimental group compared to Control group (First Questionnaire):

If the one tailed exceedance probability was less than the level of significance, the null hypothesis was rejected. Since  $0.0697 > 0.05$ , the null hypothesis could not be rejected. The conclusion was reached that there was no statistically significant difference between the two groups' mean pain at the first consultation.

It is thus assumed that both sample groups experienced a similar quality of pain at the beginning of the study. They were therefore equally matched in this respect.

Experimental group in contrast to Control group (Fourth Questionnaire):

The exceedance probability (0.07629) was again  $> 0.05$  which indicated that the null hypothesis was accepted. It was thus concluded that there was no statistically significant difference between the two groups' mean pain at the last consultation.

#### 5.1.2.2. NUMERICAL PAIN RATING SCALE 101

Experimental group as opposed to Control group (First Questionnaire):

In this test the exceedance probability (0.08795) was greater than the 0.05 level of significance. This meant that the null hypothesis could not be rejected, and thus it was stated that there was no statistically significant difference between the two groups' mean pain at the first consultation. Thus the two groups were similarly matched in terms of severity of pain.

Experimental group compared with the Control group (Fourth Questionnaire):

If the one tailed exceedance probability was greater than the level of significance, the null hypothesis was accepted. Since 0.42531 was greater than 0.05 we affirmed the null hypothesis. The conclusion was reached that there was no statistically significant difference between the two groups' mean pain as per the fourth questionnaire.

It must be remembered that if small groups of individuals are studied, a very large, absolute outcome difference must exist between the study groups before it can reach statistical significance.

The statistical test used could be an inappropriate test because the true probability of falsely concluding that there is a significant difference increases with the number of tests performed.

## 5.2. ARGUMENT

It can thus be concluded that both groups' mean pain did decrease significantly as a result of their respective interventions in terms of the quality of pain. There is therefore no differentiation between the two treatments given in this respect.

On examination of the results of the Numerical Pain Rating Scale 101, it can be seen that the mean pain of the control group did not significantly improve whilst that in the experimental group did.

It has also been determined from the above interpretation of results of both the Short-Form McGill Pain Questionnaire and the Numerical Pain Rating Scale 101 that there was no statistically significant difference between the two groups' mean pain at the first and fourth pain questionnaires.

The second and third sets of information were not used in the statistical analyses as the tests could only compare two sets of information.

A randomised clinical trial was conducted by Kokjohn, et al. (1992) involving forty-five subjects with two differing experimental interventions, that is, spinal manipulative therapy and sham spinal manipulative therapy. The reduction in plasma KDPGF<sub>2α</sub> levels in both the spinal manipulative therapy and sham treated groups that were observed is also similar to the "placebo effect" observed in this particular study. The researchers suggest further studies with more subjects studied over a longer time frame.

However, in terms of pain relief they found a statistically significant reduction in pain intensity in their experimental group compared to the control group. Unfortunately, they used a different pain intensity scale, thus making it impossible to compare that study with this one.

The weaknesses of this particular study is that the only data collected was subjective. No objective data was accumulated in the form of radio immunoassayed plasma KDPGF<sub>2α</sub>. This was due to the great expense of acquiring the equipment needed and no other local laboratory was able to assess this factor.

# CHAPTER SIX

## CONCLUSIONS AND RECOMMENDATIONS

## CONCLUSIONS

With the exception of the Numerical Pain Rating Scale 101 in the control group, when the data of the control and experimental groups were compared, it was found that both the experimental and control groups responded with a statistically significant decrease in mean scores for both pain scales. In light of this the null hypothesis was accepted.

Yet when looking at the mean pain values at the final cycle both groups experienced a significant improvement in their overall pain. Doctor reassurance and "hands on" treatment must be considered as having a direct analgesic effect on the patients. The amelioration of pain in the experimental and control groups could be as a result of the placebo effect or that both interventions work equally well.

This study suggests that spinal manipulative therapy and soft tissue therapy would be an effective and safe nonpharmacological alternative for relieving the pain and distress of primary dysmenorrhea. However, it would appear that the one intervention is no more effective than the other.

## RECOMMENDATIONS

The nonrandom, consecutive sampling, was a practical approach to selecting this subgroup of the population. A randomised, stratified sample would be more representative.



This study should be repeated using a larger sample size so that more accurate conclusions can be reached.

If this study were to be repeated it should include a means of objective assessment of the patients' response to the treatment protocol.

It would be of interest to implement a follow-up investigation at one month and four months following the last treatment to determine how effective the treatment is over a longer period.

It was necessary for the subjects to take the Short-Form McGill Pain Questionnaire and the Numerical Pain Rating Scale 101 home with them to score on the days they experienced pain. The instructions were sometimes misunderstood and the questionnaires had to be filled in again when they were handed in to the researcher. To gain further control over the questionnaires, it is recommended that they remain with the researcher in the respective subject folders and the information be obtained and filled in by the researcher personally. This would eliminate the chance of the forms being incorrectly completed.

Another study that could be recommended is to treat the subjects for symptomatic relief, that is, the treatment interventions should only be applied when the subject experiences dysmenorrhea.

A further study of interest would be to combine the chiropractic manipulation with soft tissue massage, dry needling of myofascial trigger points, a stretching routine to relieve hypertonic lumbar musculature, nutritional and exercise counselling and use of a hot water bottle. These different therapies have been recommended

individually, but it has not been determined whether the therapies coupled would be more effective than a singular application.

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# APPENDIX

## Appendix A

### PATIENT CONSENT FORM

Dear patient,

Welcome to the Technikon Natal Chiropractic Day Clinic and my research study which involves the evaluation of treatment protocols in primary dysmenorrhea (painful periods).

A thorough case history and physical examination will be undertaken together with x-rays if indicated. A gynaecological examination will be performed by a qualified gynaecologist registered with the S.A. Medical and Dental Council. This is required to establish the diagnosis of primary dysmenorrhea.

The management will consist of 16 treatments over 3 menstrual cycles. In the first cycle a Numerical Pain Rating Scale 101 must be completed on each day of pain. Thereafter the first treatment will be on the first day of the next menstrual cycle. The visits will be 2 times per week for the first 4 weeks and then once a week for the next 8 weeks. A Short-Form McGill Pain Questionnaire will be completed at the end of each cycle.

Please be as accurate as possible when completing the different questionnaires and be assured that all personal information will be treated strictly confidentially.

Your co-operation and participation is greatly appreciated.

This is to confirm that I, the undersigned, give my consent to be questioned, examined, x-rayed and treated for research purposes at Technikon Natal Chiropractic Day Clinic and agree to comply with the requests of the researcher.

_____	_____	_____
Patient's Name (printed)	Signature	Date

_____	_____	_____
Parent/Guardian (if under age 18)	Signature	Date

_____	_____	_____
Witness' Name (Printed)	Signature	Date



Appendix B

CASE HISTORY EXAMINATION FORM

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

CASE HISTORY

Patient: \_\_\_\_\_ Date # \_\_\_\_\_

File #: \_\_\_\_\_

X-ray #: \_\_\_\_\_

Age: \_\_\_\_\_ Sex: \_\_\_\_\_ Occupation: \_\_\_\_\_

Intern: \_\_\_\_\_ Signature: \_\_\_\_\_

FOR CLINICIAN'S USE ONLY

Initial visit clinician:

Signature:

Case History:

Examination:

Previous: TN  
Other

Current: TN  
Other

X-ray Studies:

Previous: TN  
Other

Current: TN  
Other

Clinical path. lab.:

Previous: TN  
Other

Current: TN  
Other

Case status:

PTT: Conditional: Signed off: Final sign out:

Recommendations:

Intern's case history

1. Source of history:
2. Chief complaint: (patient's own words)

3. Present illness:

Location

Onset

Duration

Frequency

Pain (character)

Progression

Aggravating factors

Relieving factors

Associated S & S

Previous occurrences

Past treatment and outcome

4. Other complaints:

5. Past history:

General health status

Childhood illnesses

Adult illnesses

Psychiatric illnesses

Accidents/injuries

Surgery

Hospitalizations

6. Current health status and life-style:  
Allergies

Immunizations

Screening tests

Environmental hazards  
(home, school, work)

Safety measures  
(seat belts, condoms)

Exercise and leisure

Sleep patterns

Diet

Current medication

Tobacco

Alcohol

Social drugs

7. Family history:

Immediate family:

Age

Health

Cause of death

DM

Heart disease

TB

HBP

Stroke

Kidney disease

CA

Arthritis

Anaemia

Headaches

Thyroid disease

Epilepsy

Mental illness

Alcoholism

Drug addiction

Other

8. Psychosocial history:

Home situation

Daily life

Important experiences

Religious beliefs

9. Review of systems:

General

Skin

Head

Eyes

Ears

Nose/sinuses

Mouth/throat

Neck

Breasts

Respiratory

Cardiac

Gastro-intestinal

Urinary

Genital

Vascular

Musculoskeletal

Neurologic

Haematologic

Endocrine

Psychiatric.

Appendix C

PHYSICAL EXAMINATION FORM



TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

PHYSICAL EXAMINATION

Underline abnormal findings in RED and elaborate on back of relevant page, if necessary.

Mark "NAD" if normal.

Patient: \_\_\_\_\_ File # \_\_\_\_\_

Last name

First name

Clinician: \_\_\_\_\_ Signature: \_\_\_\_\_

Intern: \_\_\_\_\_ Signature: \_\_\_\_\_

Date: \_\_\_\_\_

Height: \_\_\_\_\_ Weight: \_\_\_\_\_ Temp: \_\_\_\_\_

Rates: Heart: \_\_\_\_\_ Pulse: \_\_\_\_\_ Respiration: \_\_\_\_\_

Blood pressure: Arms: L       /       R       /

Legs: L       /       R       /

General appearance:

STANDING EXAMINATION.

Minor's sign  
Skin changes  
Posture  
    erect  
    Adam's  
"Ranges of motion:

T/L spine: Flexion: 90 Fingers to floor  
            Extension: 50  
            R.lat.flex.: 30 Fingers down leg  
            L.lat.flex.: 30 Fingers down leg  
            Rot.to R.: 35  
            Rot.to L.: 35

Flex.

L.Rot.

R.Rot.

L.lat  
flex.

R.lat.  
flex.

Ext.

/ = pain-free limitation; // = painful limitation.

Romberg's sign.  
Pronator drift.  
Trendelenburg's sign.  
Gait.

    rhythm  
    balance  
    pendulousness  
    on toes  
    on heels  
    tandem

Half squat.

Scapular winging.

Muscle tone.

Spasticity/Rigidity.

Shoulder:

skin

symmetry

ROM - glenohumeral

scapulo-thoracic

acromioclavicular

elbow

wrist

Chest measurement

inspiration

expiration

Visual acuity

Breast examination:

Inspection:

skin

size

contour

nipples

arms overhead

hands against hips

leaning forward.

Palpation:

axillary lymph nodes.

SEATED EXAMINATION.

Spinal posture

Head

scalp

skull

face

skin

Eyes

conjunctiva

sclera

eyebrows

eyelids

lacrimal gland

nasolacrimal duct

alignment

corneal reflex

ocular movement

L  
III IV VI

R  
III IV VI

visual fields

accomodation

iris

pupils

red reflex

optic disc

vessels  
general background  
macula  
vitreous  
lens

Ears:

auricle  
ear canal  
drum  
auditory acuity  
Weber test  
Rinne test

Nose:

external  
internal  
septum  
turbinates  
olfaction

Sinuses (frontal & maxillary):

tenderness  
transillumination

Mouth and pharynx:

lips  
buccal mucosa  
gums and teeth  
roof  
tongue  
inspection  
movement  
taste  
palpation  
pharynx  
inspection  
CN X

Neck:

posture  
size  
swelling  
scars  
discoloration  
hair line

ROM:

Flexion: 45 chin to larynx  
chin to sternum  
Extension: 55 forehead parallel  
to floor  
L.lat.flex: 40  
R.lat.flex: 40  
L.rot.: 70  
R.rot.: 70

Flex.

L.Rot.

R.Rot.

L.Lat.  
flex.

R.lat.  
flex.

Ext.

lymph nodes  
trachea  
thyroid  
carotid arteries (thrills, bruit)

CN V

CN VII

CN VIII (nystagmus)

CN IX

CN XI

TMJ

Inspection

ROM

deviation

Palpation

crepitus

tenderness

Neurological:

Dermatomes

C5

C6

C7

C8

T1

Tendon reflexes

biceps

triceps

brachioradialis

Muscle strength

C5

C6

C7

C8

T1

Coordination:

point-to-point

dysdiadochokinesia

Thorax:

Chest:

Inspection:

skin

shape

respiratory distress

rhythm (respiratory)

depth "

effort "

intercostal/supraclavicular retraction

Palpation:

tenderness

masses

respiratory expansion

tactile fremitus

Percussion:

lungs (posterior)

diaphragmatic excursion

kidney punch

Auscultation:

breath sounds

vesicular

bronchial

adventitious sounds

crackles (rales)

wheezes (rhonchi)

voice sounds

broncophony

whispered pectoriloquy

egophony

Cardiovascular:

auscultation (aortic murmurs)

Allen's test

SUPINE EXAMINATION

JVP

PMI

auscultation heart (L.lat.recumbent)

respiratory excursion

percussion chest (anterior)

breast palpation

The abdomen:

Inspection:

skin

umbilicus

contour

peristalsis

pulsations

hernias (umbilical/incisional)

Auscultation:

bowel sounds

bruit

Percussion:

general

liver

spleen

Palpation:

superficial reflexes

cough

light

rebound tenderness

deep

liver

spleen

kidneys

aorta

intra-/retro-abdominal wall mass

shifting dullness

fluid wave

Acute abdomen:

where pain began and now

cough

tenderness

guarding/rigidity

rebound tenderness

Rovsing's sign

psoas sign

obturator sign

cutaneous hyperaesthesia

rectal exam

Murphy's sign.

## Male genitals and hernias.

**Inspection:**

skin  
prepuce  
glans  
meatus  
nits/lice  
scrotum  
inguinal/femoral bulges

**Palpation:**

penis (tenderness/induration)  
testes  
epididymis  
inguinal canal  
femoral canal  
cremasteric reflex

**Auscultation:**

scrotal mass.

**Peripheral vasculature:**

**Inspection:**

skin  
nail beds  
pigmentation  
hair loss

**Palpation:**

pulses - radial, brachial, femoral, popliteal, post.tibial,  
dorsalis pedis  
lymph nodes - epitrochlear, femoral (horizontal & vertical)  
temperature (feet & legs)

### Manual compression test

**Retrograde filling (Trendelenburg) test**  
**Arterial insufficiency test**

**Musculoskeletal:**

## ROM

hip

```
flex.  90/120
ext.    15
abd.    45
add.    30
int rot  40
ext rot  45
```

**knee**

flex. 130  
ext. 0/15

## ankle

plantar flex	45
dorsiflex	30
inversion	30
eversion	20

leg length



Neurological:

dermatomes

L1

L2

L3

L4

L5

S1

muscle strength

hip flexion

knee extension

ankle dorsiflexion

plantar flexion

tendon reflexes

patellar

Achilles

plantar reflex

Rectal examination:

Inspection

sacrococcygeal & perianal areas

Palpation

sphincter tone

tenderness

induration

nodules

prostate

seminal vesicles

Mental status

Appearance and behaviour:

level of consciousness

posture and motor behaviour

dress, grooming, personal hygiene

facial expression

affect

Speech and language:

quantity

rate

volume

fluency

aphasia (prn)

Mood

Thought processes (logical, relevant, organized)

Memory and attention:

orientation (time, place, person)

remote memory

recent memory

new learning ability

Higher cognitive functions:

information and vocabulary (general & specialised knowledge)

abstract thinking.

Appendix D

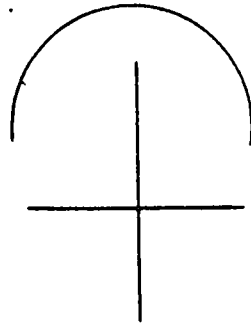
LUMBAR SPINE AND PELVIS REGIONAL EXAMINATION FORM

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

REGIONAL EXAMINATION - LOW BACK

Standing:

Minor's sign  
posture  
skin  
muscle tone  
spinous percussion  
Schober's test (6cm)  
Treadmill  
R.O.M.



Flexion 15cm from floor.

Extension 30°

R. Lat flex 35°	Fingers to knees
L. Lat flex 35°	" " "
R. rot. 30°	
L. rot. 30°	

/ painless limitation  
// painful limitation

Gait:

rhythm  
on toes (or while standing)  
on heels (or while standing)  
half-squat on one leg.

Motion Palpation:

sacro-iliacs (see below for findings)

Sitting:

Posture

Dermatomes:

T12  
L1  
L2  
L3  
L4  
L5  
S1  
S2  
S3

# Reflexes:

patellar  
Achilles  
medial hamstring.

# Reflexes:

myotomes: L. R.  
hip flex  
hip int rot  
hip ext rot  
knee ext  
knee flex  
hip abd  
hip add  
ankle dorsiflex  
ankle plantar flex  
ankle eversion  
ankle inversion  
ext. hallucis long.

tripod  
Kemp's

## MOTION PALPATION:

Jt. play		Left						Right						Jt. play	
P/A	Lat	Fle	Ext	LF	AR	PR		Fle	Ext	LF	AR	PR		P/A	Lat
							T10								
							T11								
							T12								
							L1								
							L2								
							L3								
							L4								
							L5								
					U	L	SI	U	L						

# Supine:

skin, hair, nails  
observe abdomen  
fasciculations  
abdominal reflexes  
auscultate abdomen/groin  
palpate abdomen/groin  
pulses (abd/ext)  
S.R.  
Braggard's  
bowstring  
sciatic notch  
planter reflex  
circumference (thigh, calf)

leg length:  
    actual  
    apparent  
Patrick FABER  
Gaenslen's  
gluteus max stretch  
hip medial rotation  
psoas test  
Thomas' test:  
    hip joint  
    rectus femoris.

Lateral recumbent:

S-I compression  
Ober's test  
femoral nerve stretch  
myotomes:  
    QL  
    glut.med

Prone:

gluteal skyline  
skin rolling  
iliac crest compression  
facet joint challenge  
S-I tenderness  
Erichsen's test  
Pheasant's test  
myotomes:  
    glut. max.  
trigger points:  
    QL  
    glut. med  
    glut. max  
    piriformis  
    hamstrings  
    TFL

Non-organic signs:

pin-point pain  
axial compression  
trunk rotation  
Burn's bench test  
flip test  
Hoover's test  
ankle dorsiflexion test  
pin-point pain.

## Appendix E

### NUMERICAL PAIN RATING SCALE 101

Please indicate on the line below the number between 0 and 100 that best describes the pain of your major problem when it is at its worst. A zero (0) would mean "no pain at all" and one hundred (100) would mean "pain as bad as it could be". Please only write one number.

---

Please indicate on the line below the number between 0 and 100 that best describes the pain of your major problem when it is at its least. A zero (0) would mean "no pain at all" and one hundred (100) would mean "pain as bad as it could be". Please only write one number.

---

## Appendix F

### SHORT-FORM MCGILL PAIN QUESTIONNAIRE

PATIENT'S NAME \_\_\_\_\_ DATE \_\_\_\_\_

	<u>NONE</u>	<u>MILD</u>	<u>MODERATE</u>	<u>SEVERE</u>
THROBBING	0) _____	1) _____	2) _____	3) _____
SHOOTING	0) _____	1) _____	2) _____	3) _____
STABBING	0) _____	1) _____	2) _____	3) _____
SHARP	0) _____	1) _____	2) _____	3) _____
CRAMPING	0) _____	1) _____	2) _____	3) _____
GNAWING	0) _____	1) _____	2) _____	3) _____
HOT-BURNING	0) _____	1) _____	2) _____	3) _____
ACHING	0) _____	1) _____	2) _____	3) _____
HEAVY	0) _____	1) _____	2) _____	3) _____
TENDER	0) _____	1) _____	2) _____	3) _____
SPLITTING	0) _____	1) _____	2) _____	3) _____
TIRING-EXHAUSTING	0) _____	1) _____	2) _____	3) _____
SICKENING	0) _____	1) _____	2) _____	3) _____
FEARFUL	0) _____	1) _____	2) _____	3) _____
PUNISHING-CRUEL	0) _____	1) _____	2) _____	3) _____